CASE 7.—C. G., aged 8 months, had dulness over the right apex posteriorly, with slight increase in voice and breath sounds. There was no bronchial breathing. The roentgen ray revealed partial consolidation of the right upper lobe which did not extend to the root.

The physical signs and the shadow exhibited in this case confirm the observation of Dr. Mason that bronchial voice and breath sounds do not appear until the consolidation reaches the root. This is explained by the fact that sound is conducted much better through a medium of uniform density than in one of varying density. From studies made by Dr. Mason, he believes that the consolidation of pneumonia always begins in that portion of the lung that lies close to the pleura.[1]

CASE 8.—S. S., aged 2 years, had harsh respiratory sounds over both lungs. There was dulness over the right apex, with bronchial breathing. The roentgen ray revealed complete consolidation of the right upper lobe.

CASE 9.—J. W., aged 11 months, according to the clinical diagnosis had central pneumonia. There was no bronchial breathing. The roentgen ray revealed a partial consolidation of the lower peripheral portion of the right upper lobe. This case also exemplifies the contention that what is supposed to be a central pneumonia is really peripheral at the start. The absence of bronchial breathing is explained by the fact that the inflammation has not yet reached the root of the lung.

CASE 10.—J. G., aged 8 months, had subcrepitant and sibilant râles all over the chest. There were irregular spots of dulness on the left side, and congestion of the left upper lobe. The roentgen ray revealed an infiltration, peribronchial in distribution, extending from the roots into the right upper and lower lobes. This was a case of bronchopneumonia.

In most of our cases the shadow of the heart was increased in pneumonia, especially in lobar pneumonia. The enlargement was usually most marked in the right heart.

CASE 11.—P. S., aged 16 months, had a large number of moist and crackling râles over both lungs. The roentgen ray revealed diffuse miliary infiltration through both lungs.

The cases included in this study were taken from the New York Post-Graduate Hospital and Dispensary. Dr. William H. Meyer, head of the roentgen-ray department, took and interpreted the roentgenograms. I am also indebted to Dr. Edelman for aid in collecting the cases, and to Drs. Walker and Davison of the house staff.

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1. Mason, H. H.: Lobar Pneumonia in Children; Roentgen-Ray Findings and an Explanation of Bronchial Signs, Am. Jour. Dis. Child., March, 1916, p. 188.

Control of Communicable Diseases.—*Public Health Reports*, Oct. 12, 1917, contains the report of a committee of the American Public Health Association on standard regulations concerning the control of communicable diseases. This report, which is an extensive one, gives a list of the diseases communicable and reportable, and sets forth in detail information about each, covering the infective agent, source of infection, the mode of transmission, incubation period, period of communicability and methods of control. As a guide to health authorities in the preparation of local regulations regarding these diseases, the committee has established definitions of terms such as the following: cleaning, contact, delousing, disinfection, education in personal cleanliness, fumigation, isolation, quarantine, etc. Inasmuch as the laws under which the various departments of health operate require differences in the legal phraseology of rules, etc., formal regulations have not been prepared by the committee concerning each disease.

THE LIVER AND ITS CIRRHOSES

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The liver is the central metabolic laboratory of the human body responsible for the final preparation of nutritive material for conversion into tissue-building and energizing substances. A brief review of some of its anatomic and physiologic characteristics may not be out of place. The weight of the average liver in man is 50 ounces, with a normal variation of about 10 per cent. It may be assumed, therefore, that a liver weighing more than 55 ounces is increased in size and might properly be called hypertrophic, and one weighing less than 45 ounces might be called atrophic, unless such difference could be explained by the size, above or below the normal, of the person. If the weight of the liver is an indication of its metabolic activities, the liver of the female should be larger in proportion than that of the male, made so by the necessity of taking care of herself and her unborn child. As a matter of fact, the liver of the female is only one fortieth of the body weight, while that of the male is one thirty-sixth of the body weight. The hepatic artery is relatively a small vessel and has no corresponding veins. It is supposed to nourish the liver itself and, so far as its framework, the biliary channels and the gallbladder are concerned, this is undoubtedly true; but I have not seen anatomic proof that the liver cells are nourished by the hepatic artery. It would appear that the liver cells, in the process of acting on the blood brought to them through the portal circulation, receive nourishment direct, and that there is no distinction in this respect between the blood of the hepatic artery and that of the portal vein.

The portal system is made up of the gastromesenteric veins and the splenic vein, and it should be noted that the normal splenic vein carries to the liver from one eighth to one sixth of the total quantity of portal blood. The enlarged spleen has vessels in accordance with its size. The large spleens in certain splenomegalias may have vessels the size of the superior mesenteric artery and veins. This is most important, as it indicates that removal of the spleen relieves the liver of a large load of blood and, as shown by the results of splenectomies, this diversion to the general circulation may be sufficient to relieve the subnormal liver of its overload and enable the patients to return to a fair degree of health in otherwise fatal cases. Splenectomy establishes a new principle of treatment, namely, a reduction instead of a diversion of portal circulation, as accomplished in the experimental Eck fistula and the Talma,[1] Drummond and Morison[2] operation. The stomach and rectum each have a double vascular circulation, portal and systemic. Double ligation and division of the inferior mesenteric artery and vein where they cross the left common iliac vein or just beyond at the promontory of the sacrum, where the artery takes the name of the superior rectal, would greatly reduce the portal circulation. The artery is as large as the brachial, and in two-stage operations for cancer of the rectum, experience has shown that an enormous compensatory circulation is established with the general circulation through the middle and external

1. Talma, S.: Chirurgische Oeffnung neuer Seitenbahnen für das Blut der Vena porta, Berl. klin. Wchnschr., 1898, **35**, 833-836.
2. Drummond, D., and Morison, R.: A Case of Ascites Due to Cirrhosis of the Liver Cured by Operation, Brit. Med. Jour., 1896, **2**, 728-729.

hemorrhoidal vessels. Some effect might be produced by tying the superior coronary vessels of the stomach, thus increasing the return circulation about the esophagus, but this would be relatively unimportant and might increase the tendency to gastric hemorrhage.

To the liver has been given wonderful power of regeneration. If a considerable portion of the organ is removed, it will be restored by the remaining liver cells. Such regeneration has not been given to any other organ in the human body. When the necessity for work compensation is thrown on other organs, it is by hypertrophy of preexisting elements, not cell hyperplasia, that the stress is met. The outstanding feature of the hepatic cell is that there is no differentiation of the cells. Each liver cell is exactly like every other liver cell, and each normal liver cell is fully capable of bearing its portion of the work, here again differing from every other similar organ in which groups of specialized cells are to be found.

FUNCTIONS OF THE LIVER

Our knowledge of the function of the liver is very imperfect. We have learned something from experimentation. About postmortem conditions we know a great deal, but, as the liver cannot be removed in life, and as no attempt is made to remove any considerable portion of it except for disease, which vitiates the testimony, its functions have been most difficult to ascertain. The liver has five chief functions: (1) The metabolism of carbohydrates, (2) the metabolism of proteins, (3) the metabolism of fat, (4) the production of bile, and (5) the defense against bacteria, protozoa, and toxic chemical substances.

1. The glycogenetic function of the liver is most important in the final conversion and storage of carbohydrate derivatives in a form from which energy is most readily produced. The monosaccharids might be called the body coal which heats and energizes, the ash end-product, the carbon dioxid, being carried out of the system by the ventilating function of the lungs. Sugar is a threshold body always existing in the blood but appearing in the urine only when in excess of a definite percentage.

2. The amino-acids from protein digestion, of which eighteen have been described, are carried to the liver by the portal vein and, among other changes, the nitrogen-containing portion of the molecule is there converted into urea. The conversion of the nitrogen-containing portion of the amino-acids into urea is not carried on exclusively in the liver, but the liver seems to have a greater capacity than any other tissue for this reaction. Experimental work on the development of urea is most interesting and indicates that possibly the blood urea may act as a hepatic hormone in relation to protein metabolism. Cushny[3] states that urea is not a threshold body in the blood; that is, it is always to be found in the blood and in the urine. The amino-acids are used in tissue building. They are also converted into fuel and energy-producing substances. Plummer and Kendall[4] have shown that cellular activity is sparked, so to speak, by the thyroid secretion, hyperthyroidism overenergizing this activity, and resulting in a burning up of the tissues.

3. The fat function of the liver is not well understood. We know that sugar and fat are stored temporarily in the liver, ready for immediate use. It is probable, under certain circumstances, that carbohydrates are converted into fat in the liver. It has been shown that by forced overfeeding of carbohydrates the liver of the goose may be caused to become enormously fat, constituting a well known Teutonic delicacy. Osler[5] has pointed out that the carbohydrate value of beer, although small, is sufficient when enormous quantities are drunk to cause an immense storage of fat in the human liver, and that when this fat exists in connection with a deposit of connective tissue the portal cirrhosis which follows will develop a hypertrophic instead of an atrophic liver. In acute stress, such as occurs in phosphorus and chloroform poisoning, and massive infections, the liver may undergo a most rapidly fatal fatty degeneration. Its usual response to destructive insults appears to be acute fatty degeneration. In these first three functions, namely, the metabolism of carbohydrates, proteins and fat, the liver completes a process started in the gastro-intestinal tract. In the next two — the bile and defense functions — the spleen is associated with the gastro-intestinal tract.

4. It is difficult to state whether the production of bile is purposeful or a waste which contains by-products valuable in intestinal digestion. The bile pigments are derived from destroyed red corpuscles carried to the liver, partly from the spleen. At one time, the red blood cells of the body were supposed to be completely regenerated in from seven to ten days, the estimation being based on the total amount of pigments excreted in the bile. Recent investigations, however, cause some doubt regarding the accepted opinion that the bile pigments are all derived from destroyed red cells, and indicate that the red cells have a much longer life. The latter view agrees with the known results of blood transfusion in the anemias. When enormous quantities of blood are destroyed, as in hemolytic icterus, the liver, as well as the spleen, becomes greatly enlarged—a condition that has been confused with biliary cirrhosis. Accumulating evidence, however, goes to show that, while such a liver may contain an increased amount of connective tissue, it is not necessarily related to the biliary channels, and to a very large extent, the enlargement may be looked on as a work hypertrophy with hyperplasia of the liver cells. An interesting constituent of the bile is the lipoid cholesterin; a certain amount of cholesterin is always to be found in the blood, but the amount excreted in the bile varies greatly with the condition of the patient. In the pregnant female, as reported by Aschoff, cholesterin is greatly increased; this suggests its relation to gallstones, which are four times as common in women as in men, at the same relative ages, and in 90 per cent. of the female patients with gallstones, the first symptoms are related to a pregnancy.

5. The defense function of the liver is most important. Bacteria are constantly being carried to the liver from the portal circulation, and pigments of these slaughtered bacteria are found as nonhematogenous hepatic pigment areas (Adami[6]). The spleen strains out many bacteria, as in typhoid, and protozoa, especially the plasmodium of malaria and the spirochete of syphilis; but it may be unable to destroy these organisms, and they are sent to the liver for destruction. It seems fairly clear that, at least so far as portal cirrhosis is concerned, it is related to the defense func-

3. Cushny, A. R.: The Secretion of Urine, New York, Longmans, Green & Co., 1917.
4. Plummer, H. S., and Kendall, E. C.: Personal communication to the author.
5. Osler, W., and McCrae, T.: Modern Medicine, Philadelphia, Lea & Febiger, 1914.
6. Adami, J. G.: The Principles of Pathology, Philadelphia, Lea & Febiger, 1908.

tion; the liver, losing power to absorb and eliminate diffuse poisons, attempts to encapsulate them, thus introducing the connective tissue. The spleen has been compared by Rowntree[7] to the glomeruli of the kidney, and the liver to the tubules, the one straining out the degenerated cells, micro-organisms and poisons, and the other acting on the material brought to it. The interrelated pathologic condition of the spleen and liver follows closely this interrelation of function.

CLASSIFICATION OF CIRRHOSES

The foregoing most prominent facts connected with the anatomy and physiology of the liver have been reviewed with the idea of throwing some light on the connective tissue diseases of the organ. First inaptly called cirrhosis by Laennec, on account of a tawny or yellow color which sometimes exists, cirrhosis is a term applied indefinitely and indiscriminately to almost any condition of the liver which is not understood, but in which there is an excess of connective tissue. The outstanding feature of all liver changes, the result of chronic irritation without regard to cause, is the deposit of connective tissue. This is well shown in the local cirrhotic processes which may accompany cancer, syphilis and tuberculosis of the liver.

The pathologic classifications are based on morphology, and the morphologic pictures are sometimes differently interpreted by the various authorities. To one who makes an attempt to understand the cirrhosis and who is interested in the living rather than the dead body, the pathologic descriptions are certainly far from illuminating. It is sometimes of benefit to be an amateur, in that an amateur may be able to see more clearly the larger elements, which are often lost in details; in other words, a better perspective is obtained. Generally speaking, fundamental types of cirrhoses may be distinguished; the others represent combinations or variations, rather than entities.

The two types are:

1. Portal cirrhosis, in which the chronic irritants, probably biochemical substances, are introduced through the portal vein, and in which circulatory disturbances are the most prominent clinical features, causing gastric hemorrhages, and especially ascites. Jaundice is seldom present and only as a terminal symptom.

2. Biliary cirrhosis, in which jaundice is clinically the chief symptom, ascites being absent or, if present, being a terminal condition, with the evidence pointing to an infectious cause.

In portal cirrhosis the connective tissue is introduced about the radicles of the portal vein, and in biliary cirrhosis, about the bile ducts. In both portal and biliary cirrhosis the spleen is often enlarged and has a causative relation in many cases, such as the terminal portal cirrhosis of the splenic anemias, the so-called Banti's disease.

I have never seen a case I could call Hanot's cirrhosis, and, so far as I know, this type of cirrhosis has no pathologic basis and little clinical evidence to support its existence. The large majority of cases that have taken the term of Hanot's cirrhosis are either hemolytic icterus or the ordinary type of biliary cirrhosis. As a matter of fact, hemolytic icterus, primarily a splenic disease with a work hypertrophy of the liver, has been confused with biliary cirrhosis and, as gallstones with recurring exacerbations of infections have existed in something like 60 per cent. of the cases in which we have removed the spleen for the cure of hemolytic icterus, this confusion has not been entirely without excuse. If we constantly bear in mind that, without regard to the nature of the irritant, the response in the liver is connective tissue formation, and that this may involve the whole liver or that it may exist locally, it may readily be seen where confusion has arisen. While typical portal cirrhosis, on the one hand, and typical biliary cirrhosis on the other, are well defined, atypical forms exist from mixed causes, as portal cirrhosis with secondary biliary cirrhosis from gallstone infections.

If hemolytic icterus is split off from the cirrhoses, and if it can be shown by further investigations, which our somewhat limited experience leads me to believe, that the enlargement of the liver which often exists in hemolytic icterus is a work hypertrophy and that the connective tissue formation present is not specific, much will have been accomplished in clearing up a vexed question.

Comparatively little work has been done on portal cirrhosis since the eighties, but during that period many interesting papers were written, especially by the French and English. Hilton Fagge[8] calls attention to a number of cases in which persons apparently in perfect health died suddenly from accidental causes and were found at necropsy to have had an extensive cirrhosis of the liver, suggesting some unknown factor not properly estimated. At operation I have occasionally found extensive cirrhosis of the liver unrelated to the condition which called for the operation and apparently not of immediate clinical importance.

COMPARISON OF PORTAL AND BILIARY CIRRHOSIS

It is probable that the relation of stimulants to cirrhosis of the liver, at least in this country, has been exaggerated. I have seen a considerable number of cases of portal cirrhoses in nonalcoholic young persons. Fagge shows that in Guy's Hospital for twenty-five years 14 per cent. of those dying from portal cirrhosis with ascites had complicating tuberculous peritonitis. Cheadle[9] and others have shown that, while the Laennec type of atrophic cirrhosis stands at one end of the group, representing the typical gin or hobnail cirrhosis of the liver, as many cases are to be found in which the weight of the liver is increased as there are those in which it is diminished, and the belief that such huge livers finally contract down to the Laennec type is unfounded. It is, of course, quite probable that, in the hepatitis which early accompanies the deposit of connective tissue, the liver would be somewhat enlarged before contraction. But that this is at all true of the massive livers, and especially of those containing quantities of fat, as seen in the beer drinker, cannot be credited. Our better understanding of the atrophic type of portal cirrhosis has led us to underestimate the frequency with which the cirrhotic liver is increased in size and weight. In biliary cirrhosis the liver is always enlarged. The margin of safety in the liver is very great. The patient with portal cirrhosis rarely dies from insufficiency of hepatic tissue, but death is usually brought about through changes in the circulation, and secondary complications, while in biliary cirrhosis death results from chronic jaundice and cachexia. The establishment of compensatory circulation by which blood would be passed from the portal

7. Rowntree, L. C.: Personal communication to the author.

8. Fagge, C. H.: Principles and Practice of Medicine, Philadelphia, Blakiston, 1886.

9. Cheadle, W. B.: Some Cirrhoses of the Liver, Brit. Med. Jour., 1900, **1**, 754-757; 824-826; 893-898.

vein around the liver into the general circulation, as advanced and pictured by Talma, Drummond and Morison, has given marked palliation in suitable cases. Sappey has most accurately described the venous avenues by which such compensatory circulation is brought about through nature's unaided efforts. Eck's fistula, that is, the establishment of a bypath between the portal vein and the vena cava, is purely experimental. It is of interest that in all the cases I have seen in which portal cirrhosis accompanied splenic anemia, the cirrhosis was of the atrophic type of Laennec.

In fifty-one cases of splenic anemia, in which we have removed the greatly enlarged spleen, the relief to the portal circulation has been immediate. In those cases in which cirrhosis was present, the ascites has now disappeared and several patients have lived for years, one for more than seven, in excellent health. The evidence here points to the fact that the original poison was carried to the liver from the spleen and theoretically is probably a protein derivative, filtered from the blood. But in five cases of portal cirrhosis with ascites, in which I removed the enlarged spleen, the four patients who recovered were greatly improved both as to their general condition and as to the relief of the ascites. On first thought, it seemed probable that in the removal of such a spleen I had checked the source of poisoning. On further consideration, another explanation appears possible or even probable. With the removal of the spleen, all the blood from the general circulation, which otherwise would have been sent to the liver through the splenic vein, was prevented from going there, and in this manner sufficient blood had been diverted from the liver to relieve the portal circulation. Possibly both views are more or less correct. The results in these cases should encourage us to splenectomize in suitable cases of portal cirrhosis in the future, especially when the spleen is enlarged.

Biliary cirrhosis, of the obstructed or acutely infected type, is easily understood. It exists in connection with gallstones, particularly those in the common duct, and jaundice is an early and continuous feature. In many of these cases, however, the patients are not cured by the removal of gallstones and biliary drainage. More or less permanent damage has been done to the ducts, resulting in chronic areas of infection and often in deposits of stones in the bile ducts, until thousands of such stones may be found in the liver. A second type, which is not so well understood, accompanies certain chronic biliary infections. In these it would appear that either primary hematogenous infection of the bile ducts took place or that there was an extension from a chronically infected gallbladder to the ducts. Rosenow's[10] work in revealing the specificity of bacteria, and in showing that the bacteria, usually streptococci, are to be found in the walls of the gallbladder and ducts and not in the bile, is most important. Large, soft lymphatic glands are usually to be found along the common duct and in the fissure of the liver. In chronic biliary cirrhosis, the liver is large and the walls of all the biliary ducts are extremely thick. In one instance, the lumen of the common duct was reduced at least one half by the deposit of connective tissue in the wall of the duct. Every grade of biliary cirrhosis may be found in this chronic type, which is much more liable to be accompanied by an enlarged spleen than those dependent on the more acute infections of the common duct. Not infrequently, chronic pancreatitis will be present from coincident infection. I have seen cases of this description in which there was apparently much improvement by prolonged biliary drainage to the surface, or by a cholecystogastrostomy or cholecystoduodenostomy; but as the clinical course of these patients is very chronic, I am not at all sure that cause and effect are properly related. In five cases of this type, in all of which the spleens were enlarged and the patients were more than 35 years of age, I performed splenectomy. All were improved, the jaundice was greatly reduced, though it had not entirely disappeared in any case, and the liver remained more or less enlarged. There are two possible explanations of this improvement: 1. The source of the chronic infection may have been focal at some point in the body and the toxic material resulting may have been continuously strained out in the spleen and sent to the liver, continuing the infection there. Such cases are occasionally seen in diseases that follow infectious diseases, for example, pneumonia. 2. When the spleen was removed, there was a large reduction in the necessary work to be performed by the liver.

The confusion which has arisen between biliary cirrhosis and hemolytic icterus has somewhat of a parallel in the failure to differentiate those ascites due to polyserositis (Concato's disease) and portal thrombosis from portal cirrhosis. Fagge states that for every three cases of portal cirrhosis with ascites, he saw one of ascites from polyserositis. Concato's disease may be recognized by the thick white peritoneum, by the intestines with greatly shortened mesentery clustered about the spine, and by the encasement of the liver and spleen in a thick white fibrous membrane. Free fluid is usually to be found in both pleural cavities. Pick's syndrome often exists, in which pericardial adhesions hamper the heart's action. The fact that, in some of these cases, the liver is completely encapsulated leads the uninitiated to believe that some form of cirrhosis is present; but, on excision of the strangling membrane, the liver will be found normal.

Warthin[11] has pointed out that thrombosis of the portal vein or some of its branches occasionally occurs with ascites, being a chronic malady accompanied by liver changes and splenomegalia, and usually confused with portal cirrhosis or splenic anemia.

10. Rosenow, E. C.: The Etiology of Cholecystitis and Gallstones and Their Production by the Intravenous Injection of Bacteria, Jour. Infect. Dis., 1916, **19**, 527-556.

11. Warthin, A. S.: The Relation of Thrombophlebitis of the Portal and Splenic Veins to Splenic Anemia and Banti's Disease, Internat. Clin., 1910, **4**, 189-226.

Egg Substitutes.—Cooking tests and analyses of egg substitutes were made by the laboratory of the Kansas State Board of Health, the results of which are given in the *Bulletin* for March. In the experiment of making sponge cake with no baking powder and replacing half the usual number of eggs with egg substitute, the cake stuck to the pan when done and did not have the other characteristics of true sponge cake. In another test, baking powder was used instead of eggs and egg substitute, with a like result. Averaging the price of seven egg substitutes, the analyses of which are given, the *Bulletin* says the consumer pays more per pound for the substitute than he would per pound for the dry material of hen's eggs calculated at 40 cents a dozen. The so-called substitutes are chiefly starch (70 to 90 per cent.), whereas the dried matter of eggs is essentially protein and fat. An analysis of a substitute called "eggette" showed to be false the manufacturer's assertion on the carton that the contents of the package was equivalent to twelve eggs. On the basis of protein value, the substitute was shown to be equal to only 1.7 egg; on a basis of fat, it was equal to only 0.14 egg, and in fuel value it was equal to only 2.6 eggs.